sophistries of no practical importance; on the contrary, they explain many clinical points and afford the indications for treatment. A thorough appreciation of the manner in which compensation is established and maintained, together with a recognition of the difficulty of restoring compensation when it has been lost, can only be obtained from a perfect knowledge of the cardiac mechanism and the manner in which it is liable to suffer under various abnormal conditions.

It is a fact that in many instances aortic lesions are regarded too lightly at their inception, and a patient allowed to resume active duties before there has been time for sufficient compensatory hypertrophy to develop, with the result that the mitral orifice becomes incompetent, and this is much harder to overcome when associated with an aortic lesion than when it occurs alone. Clinicians will generally admit this to be the case, and it is not difficult to find the explanation. Cases of muscular mitral incompetence can be relieved only by resting the heart and improving its nutrition, and success in the latter direction depends upon the possibility of restoring and maintaining an adequate circulation through the myocardium itself. To attain this end a rise in aortic pressure must be obtained, because this is one of the chief factors in determining the flow of blood through the coronary arteries; but the constricted aortic orifice, by diminishing the volume of blood entering the aorta or the incompetent valves which allow the blood to escape, present obstacles to the production and maintenance of normal aortic blood pressure which do not exist in the uncomplicated mitral lesion.

RECENT ADVANCES IN ANATOMY AND PATHOLOGY OF THE HEART FROM A CLINICAL POINT OF VIEW.[1]

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In a period of transition, such as that which at present involves our knowledge about heart disease, it is difficult to appraise properly what is and what is not important. To sum up the change in viewpoint which the subject is undergoing, it may be said that while up to five or six years ago we concerned ourselves with valvular lesions, and devoted all our energies to diagnosticating them from the point of view of sound and murmurs, and to a less extent from that of dilatation, as evidenced by percussion, palpation, and transillumination, now we have come to regard the heart as a whole, and to lay more stress on its working muscular portions and less on its automatically acting valves. It is not meant that valvular defects, either acute or chronic, have assumed a subsidiary place in our study, but that muscle function as such has been raised to a position of such importance that phenomena, which formerly were unrecognized clinically, are now capable of furnishing us with much valuable information both from a diagnostic, prognostic, and also, as many are hoping, from a therapeutic viewpoint. If we dismiss valvular diseases, therefore, from this survey, it is not because we consider them unimportant, but because recent advances have not been in their direction.

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[1] Read before the New York Academy of Medicine, April 20, 1911.

About thirty years ago Englemann and Gaskell, by extensive experiment, first placed our views about the functions of the heart muscle on a more exact basis. As a result of their work we have now come to speak of the heart muscle as possessing the functions of irritability, contractility, rhythmicity, and conductivity (in Engleman's phraseology, the bathmotropic, inotropic, chronotropic, and dromotropic functions of the heart muscle). We do best when we investigate heart muscle and heart disease under these separate heads, since frequently one patient suffers from a defect of only one of these functions, while another suffers from a combination of two or more, either as the ultimate result of a valvular lesion or, what is coming to be recognized as almost as common, a functional disturbance without the existence of a valvular defect. From a clinical point of view much has been added to our knowledge of rhythmicity and conductivity, but scarcely anything to that of irritability or contractility. As I shall point out, even that function of which we know most, namely, conductivity, leaves many points quite unexplained.

When we investigate the anatomical facts which underlie these functions of heart muscle, we are obliged to subdivide them into those which are muscular and those which are nervous, and to assign to each its proper domain.

The teaching that nerve fibrils surround and themselves interlace about the muscle fibers of the main body of the ventricular musculature is already old and accepted. Much has been added to our knowledge of the location of ganglia within the heart both of man and the lower animals. More recently, Tawara,[2] Wilson,[3] Engel,[4] and others have demonstrated the presence of nerve fibrils in the same sense about the auriculoventricular bundle, described by Kent,[5] His,[6] Braeunig,[7] Retzer,[8] Humblet,[9] Tawara,[10] and in

[2] Das Reitzleitungs system des Säugetierherzens, G. Fischer, Jena, 1906.

[3] Proc. Roy. Soc., 1909, Series B, vol. lxxxi, pp. 151 to 164.

[4] Ziegler's Beiträge, 1910, Band xlviii, 499 to 526.

[5] Jour. of Physiol., 1893, xiv, 233 to 254.

[6] Arbeit. aus der med. Klinik zu Leipzig, Leipzig, 1893, 14 to 50.

[7] Arch. f Anat. u. Physiol., Anat. Abt., 1904.

[8] Arch. Intern. de Physiol., 1904, i, 278 to 286.

[9] Ibid.

[10] Loc. cit.

some species have even shown that ganglion cells can be found far down in the ventricle in close union with the ventricular branches. These anatomical facts are known, but as yet nothing has been added to our knowledge of their function, and, more especially, no clinical abnormality has been correlated with a defect or lesion involving them. Furthermore, although a considerable literature has been accumulated in relation to the extrinsic nerves and central ganglia, both anatomical and physiological, it must be confessed that, in spite of the many facts collected in regard to the action of the vagi, the accelerators, and the depressor nerves, clinical knowledge of them is very deficient, and in many respects absent. Finally, it may be pointed out that there exists practically no investigation which relates the paths and distribution of the extrinsic with the system of intrinsic cardiac nerves.

The most that can be said is that certain manipulations of the extrinsic nerves produce certain effects and that the calling forth of these effects are very limited in number and are not constant phenomena. Here then is a large though difficult field for future investigation, and it would seem that, until much more knowledge along these lines is at hand, a myogenic and a neurogenic doctrine may be useful as points of view from which to investigate, but these doctrines should certainly not furnish a basis for the erection of hostile camps, from both of which polemics constantly keep pouring forth.

The study of the muscular architecture of the heart has brought us a large body of facts which have proved clinically extremely useful, even though this study has been concerned only with the functions of conduction and rhythm. Great advance was made after Gaskell and Englemann gave utterance to the doctrine that the propagation of an impulse once formed was conducted to other and appropriate locations in the heart by means of the heart muscle, and that in the muscle as such resided this function of conduction. Unfortunately no muscular connection between auricles and ventricles was known in the eighties, and it was not until 1893 that anatomical confirmation was given this theory by Kent[11] and His, Jr.,[12] who found what is now recognized as the auriculoventricular bundle. Braeunig,[13] Retzer,[14] and Humblet[15] confirmed the existence of this structure, but it remained for Aschoff and Tawara[16] to find its beginning in the lower interauricular septum and its ending at the venous base of the heart, in the papillary muscles. At the beginning of this conduction system and where the auricles join it, Tawara described a structure which is now called by his name, the node of Tawara, quite different in its anatomy from the rest of the heart muscle.

[11] Loc. cit.

[12] Loc. cit.

[13] Loc. cit.

[14] Loc. cit.

[15] Loc. cit.

[16] Loc. cit.

A point which should be clearly made is that no part of this A-V system should be called embryonic or an embryonic remains. Whoever has examined embryo hearts will know that it has no similarity with embryonal cardiac muscle. And Keith[17] has said: "If by embryonic is meant that they resemble the cardiac fibers of the heart of the embryo, then nothing could be further from the truth; if by embryonic is meant the fact that only the superficial stratum of the fibers is striated, the interior being undifferentiated cell substance, then in this feature they are embryonic. They are cardiac fibers which have specialized in a peculiar direction; their fibrillar structure is especially distinct."

To return, however, to the node of Tawara. Much has been written about its function, both as a pacemaker for the heart in abnormal conditions, a function once ascribed to it by Mackenzie, but now entirely abandoned by him, and as a site for the formation of so called atrioventricular extrasystoles. Actually there is known to us only one series of experiments which has been able to ascribe a definite function to this structure, and this is contained in a paper by H. E. Hering,[18] in which he maintains that the node of Tawara delays conduction. He ascertained this fact by stimulating below the level of the node and calculating the conduction time from the instant of stimulation until there occurred contractions of the auricle on the one hand and the ventricle on the other, and from these making the necessary deductions. Further than this nothing is known about the node. Aside from the node and bundle, the demonstration of nerve trunks, nerve fibers, and nerve ganglia in the conduction system, and a demonstration of the constant union of the A-V node with the auricles—a point that was necessary to complete the knowledge of the anatomical path and to show that normal automatism was a function which need not be ascribed to the auricle any more than to the ventricle—complete our anatomical knowledge about the auriculoventricular conduction system.

Knowledge about rhythmicity was augmented by the description of a node similar in structure to Tawara's in the wall of the right auricle, by Keith and Flack.[19] It begins at the junction of the superior vena cava and the upper border of the right auricular appendix and extends from there downward a varying distance in the direction of the sulcus terminalis. Structures similar to this are found at other sites, and may ultimately be shown to have functions not now ascribed to them, but we will at this time neglect them. The sino-auricular node has communications with auricular musculature in every direction. Its site and microscopic anatomy have been confirmed by Koch[20] and other observers. That rhythmicity—stimulus production—has its seat in this node and that it acts under normal conditions as the pacemaker of the heart, was a function originally claimed for it by Keith. That this function resided in this region had been presumed by McWilliam,[21] Adam,[22] Langendorff and Lehmann,[23] Erlanger,[24] and Hering.[25] Since then, except only the negative experiments of Jaeger[26] and Magnus-Alsleben,[27] investigations by Lewis,[28] Lewis and Oppenheimer,[29] Wybauw,[30] Cohn and Kessel,[31] have tended to support and confirm this view.

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[17] Lancet, 1906, ii, 359 to 364.

[18] Pflüger's Arch., 1910, cxxxi, 572 to 581.

[19] Jour. Anat. and Physiol., 1907, xli, 172 to 189.

I have said that the sino-auricular node communicates at its entire circumference with the auricular muscle. Thorel[32] imagined, however, that a communication between it and the A-V node was necessary for physiological reasons. The assumption was not, however, required by what is known of muscular conduction, and was not borne out by many other observers who failed to substantiate his anatomical findings. From the point of view of anatomy, therefore, counting more especially from the time of Braeunig and Retzer, the last seven years have put us in possession of the sino-auricular node, described by Keith and Flack, in which most probably resides the pacemaking function of the heart; and of the auriculoventricular system, through which stimuli pass from the auricles to the ventricles, so maintaining correlated contraction between these two pairs of cavities.

When we turn to that portion of the pathological anatomy of the heart which deals with the four divisions of its function above named, we find again, and naturally, that investigation has been confined only to the functions of rhythmicity and conductivity. It may be that the other functions have no anatomical substratum, and it may also be that all the functional deficiencies in these two, that is, rhythmicity and conductivity, cannot be explained anatomically, but the fact remains that anatomical explanations are at hand only for these two.

Sixty per cent. of all cases of irregularity of the heart fall under the heading of the so-called pulsus irregularis perpetuus, known also as the completely irregular pulse, nodal rhythm, and indeed by a variety of other names.

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[20] Deutsch. med. Woch., 1909, No. 10, pp. 429 to 432.

[21] Jour. of Physiol., 1888, ix, 167.

[22] Pflüger's Arch., 1906, Band cxi, 607 to 619.

[23] Ibid., Band cxii, 352 to 360.

[24] Amer. Jour. of Physiol., 1907, xix, 125 to 174.

[25] Pflüger's Arch., 1907, cxvi, 143 to 158; Münch. med. Woch., 1909, lvi, 845.

[26] Deutsch. Arch. f. klin. Med., 1910, Band c, 1 to 11.

[27] Arch. f. exp. Path. u. Pharmak., 1911, lxiv, 228 to 243.

[28] Heart, 1910, ii, 23 to 47.

[29] Ibid., 147 to 169.

[30] Arch. de Physiol., x, i. Quoted from Münch. med. Woch., 1911, No. 4, p. 215.

[31] Arch. Intern. Med., 1911, vii, 226 to 230.

[32] Münch. med Woch., 1909, No. 42, p. 2159.

The history of its explanation is an interesting example of the history of anatomical and physiological advance. When Mackenzie[33] first described the condition, he believed it to be due to auricular dilatation and paralysis. At that time the anatomy of the A-V system was practically unknown. But paralysis meant atrophy, and Mackenzie soon saw that cases with a pulsus irregularis perpetuus might actually show hypertrophy of the right auricle. Then it happened that the A-V node, supposed to be embryonal in structure and therefore of pacemaking fiber, was credited with the function of stimulus production. Lying between auricles and ventricles, and almost equidistant between them, impulses starting here and going in both directions would arrive at and set in motion both pairs of cavities at the same time. The explanation was simple; it accounted for the auricular hypertrophy, for the auricle kept beating; it accounted for the absence of the A-wave in the jugular pulse, since auricles and ventricles would be beating synchronously; it even accounted for the increased auricular rate often found. At the same time, to lend weight to the theory, Keith found a variety of lesions in and about the A-V node and bundle; these formed the factors which were alleged to irritate the A-V node to increase its activity.

The explanation neglected, however, the doctrine that automatically beating heart portions beat regularly and not irregularly, and herein lay the weakness of the explanation, a weakness that had been pointed out as an *a priori* objection. The explanation was, however, held until the fall of 1909, when Lewis,[34] and Rothberger and Winterberg[35] were able to demonstrate conclusively that the irregularity was due to auricular fibrillation, a theory which explains auricular hypertrophy, an absent A-wave from the jugular pulse, and by comparing the experimental with the clinical tracings, the perpetual irregularity as well.

Now the anatomical interest shifted from the A-V system to a study of the right auricle. Schönberg,[36] Hedinger,[37] Koch,[38] have reported histological studies in this region, while many others are still in progress. The first studies of Schönberg and Hedinger may be briefly dismissed; they concerned themselves with lymphatic infiltration in a strand of auricular muscle which ran from behind and below, upward and forward on the superior vena cava. Wenckebach has supposed that if stimuli were produced at the entrance of the great vein to the auricle, over this pathway the stimuli must be conducted to the auricle. This notion is, of course, no longer held, and therefore lesions in this area no longer deserve the attention they once received, nor have they the significance once attached to them. In the sino-auricular node, however, Koch and others have found subendocardial fatty infiltration, small hemorrhages, subacute inflammatory changes, injuries to the nerves and ganglia by connective-tissue growth, occasional lesions in the afferent vessels, and more or less diffuse connective-tissue proliferation. These lesions cause destruction of the structures of the sino-auricular node. Such lesions, not by any means uniform, nor all found in any one heart, are designated as lesions sufficient to injure the pacemaking function of the node and so induce either fibrillation directly, or, in any event, to remove the controlling influence of the sinus, in such a fashion as indirectly to permit fibrillation to ensue. As I shall point out later, in speaking of the lesions of the A-V system, enough cases have not yet been examined nor sufficiently examined to make it advisable to correlate definitely anatomical lesions with functional change; in short, to make the anatomical changes sufficient explanation for the onset of fibrillation. Furthermore, it must be borne in mind that perpetual irregularities are of varying rates, now slow, now fast, and that there are cases in which the rate changes several times during the progress of the case. Therefore, while these anatomical advances and findings are valuable, interesting, and possibly final, enough is not known to consider them conclusive, and harm rather than good may be done by accepting them now.

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[33] Brit. Med. Jour., 1904, i, 529.

[34] Heart, 1910, i, 306 to 372.

[35] Pflüger's Arch., 1910, Band cxxxi, 387 to 407.

[36] Frankfürter Zeitschr. f. Path., 1908, ii, 153 to 180; ibid., 462 to 485.

[37] Ibid., 1910, v, 296 to 322.

[38] Berl. klin. Woch., 1910, 1108 to 1112.

I turn now to what is known of lesions of the auriculoventricular system and their clinical manifestations. So far isolated lesions involving either the auriculonodal junction or the auriculoventricular node itself are not known. Lesions have been described here, such as subacute, acute, and gummatous inflammations, but these are correlated with no clinical manifestations and require no further notice. When we go on to the main stem, however, a variety of lesions are noted. These may be divided into those which form complete transverse lesions of the bundle, in the same sense that there are complete transverse lesions of the spinal cord, and incomplete transverse lesions. This latter group contains both disseminated lesions and lesions which do not completely divide the bundle.

In the group of complete transverse lesions are enumerated tumors, both benign and malignant, metastases or infiltrations; inflammations, usually syphilitic, among which the calcified gumma plays an important part; and degeneration due to vascular disturbance, either thrombotic or embolic, or endarteritic. These complete lesions, so far as I know, all cause complete auriculoventricular dissociation, and may or may not be associated with the Adams-Stokes syndrome.

In the second group are those cases in which the lesion does not divide the A-V bundle completely. In these there are found the products of chronic inflammation, firm old connective-tissue infiltration of the main stem, and infiltrations with rather large quantities of fat tissue. A special type consists of the presence in the bundle of large blood sinuses which have materially reduced the diameter of the bundle. It must be confessed that in the group of incomplete transverse lesion, cases are found where the auriculo-ventricular dissociation is complete, and others where it is partial. Whether the amount of injury alone is responsible for the varying degree of block or whether additional factors related to nerve control, nerve destruction, or nerve tone, are here involved, more expecially in the cases of complete dissociation, cannot now be properly appraised.

A third group[39] of cases must be added in which there are marked lesions of the bundle, but in which, clinically, all types of heartblock are demonstrated, from lengthened A-C intervals to complete dissociation, changing from one form to another in rather a startling fashion. To account for such cases, it has been supposed that the block has been from time to time increased and then diminished by the presence of inflammatory processes which undergo exacerbations and then subside, and which in their organization tend effectually ultimately to destroy the bundle.

In addition to these three groups is a fourth, in which heart block has existed clinically for years, as in Krumbhaar's[40] case, in which a lesion commensurate with the clinical findings cannot be demonstrated. In fact, it can be shown that the hearts in such cases contain lesions less in amount than are observed in hearts known to present no manifestation of disturbed conductivity during life.

Finally, there are a few cases in a fifth group in which, through inflammation or otherwise, one or other of the ventricular branches of the A-V system have been found cut off from the main stem. These cases reported by Eppinger,[41] aside from rather vague but certainly distinct clinical manifestations, have been diagnosticated electrically, and are of great clinical interest.

We have now summed up lesions in the heart involving the sino-auricular node and having their expression possibly in the cases of perpetual irregularity. We have also shown what lesions occur in the auriculoventricular system, and have shown their relation to heart block. We have not considered a number of the other irregularities, also chronotropic functions, expressed in extrasystoles and in praoxysmal tachycardia.

Whether chronic and acute interstitial myocarditis plays a role in the production more especially of ventricular extrasystoles is not known. The assumption is held, nevertheless, and the cases are called cases of cardiosclerosis. It is, however, a fact that there is no definite correlation known as yet between abnormal structure and abnormal function in any of these cases, nor are the limits of structural change in general convertible into the extent of functional deficiency. An attempt in this direction has been made by Monckeberg[42] when he considered certain cases of sudden death due to fatty degeneration of the A-V bundle, but his findings have been both confirmed and denied by Engel,[43] and their status is not secure.

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[39] Quart. Jour. Med., 1910, iii, 126 to 152.

[40] Arch. Int. Med., 1910, v, 583 to 596.

[41] Zeitschr. f. klin. Med., 1910, Band lxxi, 157 to 164.

Aside from the functions mentioned, little is known. Much is left to be investigated, notably in the domain of irritability and of contractility of heart muscle, expecially in acute infections and in chronic degeneration. The string galvanometer, which produces electrocardiograms, at first seemed destined to help in directing these investigations, and likely much remains for it to do. From the point of view of these functions, anatomy has at this time little in the way of explanation to offer.

In these days, when we are set about by tracings made in a variety of ways, by instruments that detect numerous different physiological activities, and by the effort to understand the meanings of the tracings once obtained, one must insist and insist strongly that all this is but machinery, nothing but a means to an end. It is as aids in understanding the heart in health and disease that this cumbrous apparatus is called into being. The tracings which they yield will be exceedingly barren, unless the effort to interpret them from the standpoint of anatomy and of physiology, both normal and abnormal, is constantly made.

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[42] Untersuchungen über das Atrioventrikular bundel im menschlichen Herzen, Fischer, Jena, 1910.

[43] Loc cit.

REVIEWS

JOINT TUBERCULOSIS. By LEONARD W. ELY, M.D., Consulting Orthopedist to the County Hospital, Attending Orthopedist to the Children's Hospital, Denver, Colorado; formerly Surgeon to the Sea Breeze Hospital, and Consulting Orthopedist to the Roosevelt Hospital, New York City. Pp. 243; 72 illustrations and a colored frontispiece. New York: William Wood & Co., 1911.

THIS is a very interesting book. It was begun by the author as a clinical study, but finding this inadequate to explain the phenomena of joint tuberculosis, he turned to the pathology, and, from an examination of 72 specimens from 64 patients, has come to some conclusions which must be regarded as of far-reaching importance if their truth be substantiated by further observation.

He found both osseous and synovial lesions as the primary focus, the latter very rare in children, but much more common in adults. Obliterating endarteritis was observed as a frequent characteristic in these tuberculous lesions.

The localization of tuberculous foci in the epiphyses of children he explains solely by the presence there of red marrow. Where red marrow exists there is a predisposition to the development of tuberculous lesions; and where there is no red marrow the bone is almost immune to infection by tubercle bacilli. Other explanations, he says, such as the lack of terminal anastomoses predisposing to embolism, do not explain. If red marrow can be converted into yellow marrow, tuberculous lesions will heal. He regards lymphocytic aggregations around tubercle bacilli not as the *result*, but as the *cause* of the tuberculous focus; lymphocytes, he contends, as well as connective tissues which contain epithelial, epithelioid, or lymphoid cells, are the natural food of tubercle bacilli; where these elements are not already present, tubercle bacilli do not thrive. He doubts the existence of primary fascial tuberculosis because there exist in fascia no cellular elements suitable for the nourishment of tubercle bacilli.

From these pathological inferences—for at present they can scarcely be said to be more—the author finds a ready explanation of and indication for treatment. *Rest* is the main therapeutic indication, and it succeeds not because of either traction or fixation, which